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OUTBREAK OF INFLUENZA A IN AN ONTARIO NURSING HOME — JANUARY 1997

On 2 January 1997, the Wellington-Dufferin-Guelph Health Unit was alerted to a possible outbreak of influenza in a nursing home. Symptoms for the first case began on 1 January. At the time the Health Unit was alerted, 15 residents had developed symptoms including muscle aches, malaise, cough, sore throat, and fever. Nasal swabs were taken from three patients; one positive result for influenza A was obtained by rapid testing (enzyme-immunoassay). This single confirmation was sufficient to declare the occurrence of an outbreak.

The institution responded by initiating an amantadine regimen for all residents. Asymptomatic residents were put on a 2-week daily-dose regimen, while those already ill were offered a 5-day regimen in order to reduce the severity of illness. Residents who became ill after amantadine was initiated were transferred to the 5-day regimen. Daily dosage was set on the basis of creatinine levels. Residents with normal creatinine levels (< 120) received a daily dose of 100 mg of amantadine. Local supplies of amantadine were limited; therefore, amantadine was obtained from out-of-town sources. The first dose was administered on 2 January.

All residents as well as staff who had not yet received influenza vaccine in the fall were offered immunization concurrent with the outbreak. Other precautions included the isolation of identified cases, a halt to all activities between wards, and the exclusion of all visitors.

The outbreak was tracked through line listings of cases which were maintained for each floor. The onset date of the last resident to become ill was 11 January 1997.

Outbreak Description

Residents were considered cases if they were ill for > 2 days with any of the following symptoms not explained by a pre-existing condition: fever > 38° C, nasal congestion, cough or sore throat, muscle aches, or lethargy. Staff were identified as cases if they had at least three of the above symptoms lasting > 2 days.

Forty-one percent (71 of 172) of residents met the case definition. Of these, 12 were male and 59 female. The average age was 87 years (range: 61 to 103). Six cases died of respiratory illness during the outbreak (case fatality: 8.5%).

Ninety percent (171 of 189) of staff members responded to a survey conducted immediately following the outbreak to determine vaccine coverage and influenza attack rates among the staff. Fifty-one staff members reported an influenza-like illness; however, 11 were not considered cases since their symptoms lasted for only 1 or 2 days. Of the 95 direct-care staff, 26% (25) became ill with influenza-like symptoms lasting > 2 days between 1 December 1996 and 16 January 1997. Of the 76 non-direct-care staff, 20% (15) were ill. During the outbreak period, there were 18 direct-care and two non-direct-care staff who became ill. This included staff who reported onset 2 days before the first ill resident and 2 days after the last ill resident.

Influenza A attack rates for residents varied among wards from 22.4% in Unit 1 to 84.1% in Unit 4 ($x^2 = 45.7$, df = 3, $p < 0.001$) (Table 1). Residents on Unit 1 were more likely to be ambulatory and relatively independent. Unit 4, with the highest attack rate, was one of two wards with residents having the highest personal-care needs.

Table 1
Influenza A attack rates and vaccine coverage for residents of an Ontario nursing home, by ward

Unit	Vaccine coverage	Attack rate
1	88.1% (59/67)	22.4% (15/67)
2	96.6% (28/29)	31.0% (9/29)
3	84.4% (27/32)	31.3% (10/32)
4	79.5% (35/44)	84.1% (37/44)

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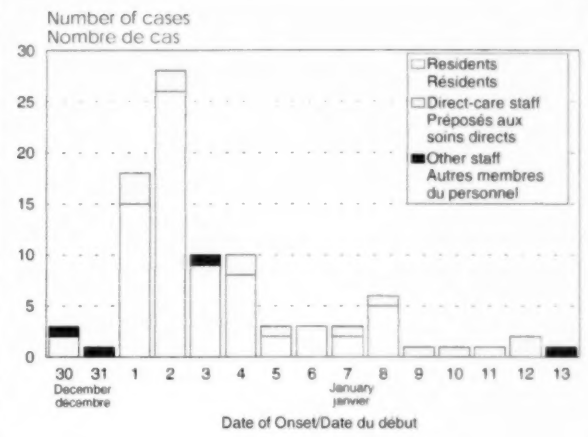
Figure 1
Epidemic curve – influenza A outbreak in an Ontario nursing home, January 1997



Overall, 86.6% (149 of 172) of nursing-home residents had received influenza vaccine in the fall. The recommended target for vaccine coverage of nursing-home residents is 90%[1]. Differences in the vaccine coverage rates among the units were not statistically significant ($x^2 = 4.6$, df = 3, p = 0.2). Note that Unit 4 with the lowest vaccine coverage rate (79.5%) also experienced the highest attack rate (84.1%).

The immunization rate of direct-care staff in the home was 20% (19 of 95) and 22% (16 of 73) for the non-direct-care staff. Direct-care staff represent one-third of the nursing home community. The vaccine coverage for the nursing home, direct-care staff and residents combined, was insufficient to prevent an outbreak.

Table 2 presents influenza A attack rates among residents and staff by immunization status.

Discussion

These results raise some questions concerning the effectiveness of the vaccine; so many of the residents as well as staff became ill with influenza despite having been vaccinated in the fall. A closer look at the vaccine and outbreak data suggests other factors that may have influenced the rate of influenza occurrence.

The high attack rate on Unit 4 may be attributed to greater potential exposure among Unit 4 residents to direct-care staff carrying the virus, and potentially poorer responsiveness to the influenza vaccine. Most of the 1 January cases (13) occurred on Unit 4; Unit 1 and 3 also identified an influenza case each on the same day.

Table 2
Influenza A attack rates for residents and staff of an Ontario nursing home, by vaccine status

Group	Vaccinated	Not vaccinated	x^2	p
Residents	38% (57/149)	52% (12/23)	1.6	0.2
Staff (1 Dec to 16 Jan)	26% (9/35)	23% (31/136)	0.3	0.7
Staff during the outbreak (30 Dec to 13 Jan)*	15% (4/27)*	15% (16/106)*	—	—

* Staff reportedly ill **before** the outbreak are excluded from the denominators.

One-half of the reported influenza illnesses among staff were not a part of the institution's outbreak (i.e. 20 of 40 episodes occurred outside of the period of communicability for the outbreak). Because there is no obvious epidemiologic link to the outbreak, it is suspect whether these other reported episodes were truly influenza or, in fact, some other infectious illness with a similar presentation.

Because the level of immunity for the frail elderly is likely to taper off over time following vaccination for influenza, it is generally recommended that the vaccine be administered to nursing-home residents during the later half of October to the middle of November, closer to the months of December and January when influenza is most likely to occur[2]. In this institution, 79% (118 of 149) of residents vaccinated, received their vaccine before 15 October 1996. The attack rate for those vaccinated before 15 October was 47.5% (56 of 118) compared to 9.7% (3 of 31) among those vaccinated after 15 October (RR = 4.9, 95% CI = 1.65 to 14.6). In other words, residents vaccinated before 15 October were five times more likely to develop influenza A compared to residents vaccinated after 15 October. The vaccine efficacy for later vaccination is 79.6% (95% CI = 39.2 to 93.2).

Most staff who received their vaccine through the nursing home would have been given the vaccine in early October; 63% (20 of 32) reported receiving their vaccine in October 1996. Thirty-five percent (7 of 20) of those who were vaccinated in October or earlier reportedly became ill with the flu, compared with 25% (3 of 12) vaccinated after October. The differences are not significant (OR = 1.4).

The use of an amantadine regimen at the onset of the outbreak had a dramatic effect, with the number of new cases after 2 January declining rapidly. Although inconclusive (i.e. other control measures as well as a decrease in the number of susceptible residents may have also influenced the decline), this pattern is consistent with the expected response to amantadine.

Furthermore, those who became ill after the first dose of amantadine were not as ill as those who became ill initially. The duration of illness was shortened on average by 2 days. The average duration of illness was 6.5 days for the 39 residents with onset date 2 January or earlier, compared with 4.3 days for the 29 residents with later onset (t = 3.5, df = 66, p = 0.001).

Conclusions and Recommendations

The outbreak among residents lasted 11 days with the majority of cases (41 of 71) occurring on days 1 and 2. The quick response of the nursing home staff in initiating control measures was effective in limiting the outbreak. This experience points out a need for the following:

- higher levels of immunization coverage among direct-care staff, and a uniformly high coverage for residents in all wards
- a campaign that occurs in the late fall (late October to mid-November) unless there is evidence of an early influenza outbreak in the community at large
- standing orders for amantadine in response to influenza A, and a ready supply to initiate a response as soon as an outbreak is declared.

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Source: *S Isaacs, MSc, BScN, Field Epidemiology Training Program, LCDC, Ottawa; C Dickinson, RN, Wellington; G Brimmer, RN, DPHN, Wellington-Dufferin-Guelph Health Unit, Fergus, ON.*

International Notes

DECREASING INCIDENCE OF PERINATAL GROUP B STREPTOCOCCAL DISEASE — UNITED STATES, 1993-1995

Group B streptococcal (GBS) infections are the leading cause of bacterial disease and death among newborns in the United States and an important cause of morbidity among peripartum women and non-pregnant adults with chronic medical conditions. Disease in infants usually presents as sepsis, pneumonia, or meningitis but also may include cellulitis or osteomyelitis[1]. In 1990, GBS infections caused an estimated 7,600 serious illnesses and 310 deaths among US infants aged ≤ 90 days; infections among infants aged < 7 days (i.e. early-onset disease) accounted for approximately 80% of these illnesses[2]. To determine the incidence of GBS disease during 1993 to 1995, the United States Centers for Disease Control and Prevention (CDC) conducted surveillance for this disease in an aggregate population of 12.5 million persons with 190,000 annual live-born infants. This report summarizes the findings of surveillance in this population, which indicate that a statistically significant decline in the incidence of early-onset GBS disease occurred in some surveillance areas.

Surveillance was conducted in the three-county San Francisco Bay area, California; four urban counties in Tennessee; the eight-county metropolitan area of Atlanta, Georgia; and the entire state of Maryland. At biweekly intervals, surveillance personnel requested standardized reports of cases of invasive GBS disease from contacts in each laboratory that served acute-care hospitals within specified surveillance areas. Periodic audits of all laboratories were conducted to validate completeness of reporting. A case of invasive GBS disease was defined as isolation of group B streptococci from a normally sterile site (e.g. blood or cerebrospinal fluid) from a resident of an area under surveillance. Cases were categorized as early-onset and late-onset (illness onset at age 7 to 90 days). To calculate the incidence of neonatal GBS disease for the surveillance areas, the number of live-born infants for 1993 to 1995 was obtained from the respective state health departments or from CDC's National Center for Health Statistics. Race-specific data are presented only for blacks and whites because numbers for other racial and ethnic groups were too small for meaningful analysis.

During 1993 to 1995, a total of 3,023 cases of invasive GBS disease were reported from the surveillance areas; 1,071 (35%) cases occurred among newborns aged < 90 days. Of the 1,071 cases among newborns, 520 (49%) occurred among blacks, and 478 (45%) among whites. Approximately three-fourths (822 [77%]) of cases were early-onset disease. Bacteremia (89%) and meningitis with or without bacteremia (10%) were the most common types of neonatal disease.

The case-fatality rates were 4.0% for neonatal disease, 4.5% for early-onset disease, and 2.4% for late-onset disease. Of the 708 (66%) infants for whom data about gestational age were available, 85 (12%) were born at < 34 weeks gestation, 65 (9%) at 34 to 36 weeks, and 558 (79%) at ≥ 37 weeks. In comparison, of all infants born in the United States in 1993, 89% were born at ≥ 37 weeks[3]. Preterm infants were more likely to die than those born at ≥ 37 weeks (23 [16%] of 148 versus 11 [2%] of 553).

During 1993 to 1995, the overall annual incidence of early-onset GBS disease in the surveillance areas declined 24%, from 1.7 cases per 1,000 live-born infants in 1993 to 1.3 per 1,000 in 1995 (Figure 1). The race-specific incidence rate declined 27% for black newborns and 18% for white newborns. The decline in the overall incidence primarily reflected changes in rates for newborns in Maryland and San Francisco: in both of these sites, the rate of early-onset GBS disease decreased 43%, from 1.4 per 1,000 in 1993 to 0.8 per 1,000 in 1995. No significant decline occurred in Tennessee or Atlanta during this period. In 1995, the rate varied by geographic location, ranging from 0.8 (Maryland and San Francisco) to 1.9 (Atlanta), and race-specific rates were higher for black newborns (1.8) than for white newborns (1.2). During this same period, the rates of late-onset neonatal GBS disease (0.5 in 1993 and in 1995) and of GBS disease for adults remained stable.

MMWR Editorial Note

Since the mid-1980s, multiple studies have demonstrated consistently that early-onset GBS disease can be prevented by targeted use of antimicrobial prophylaxis after onset of labor or

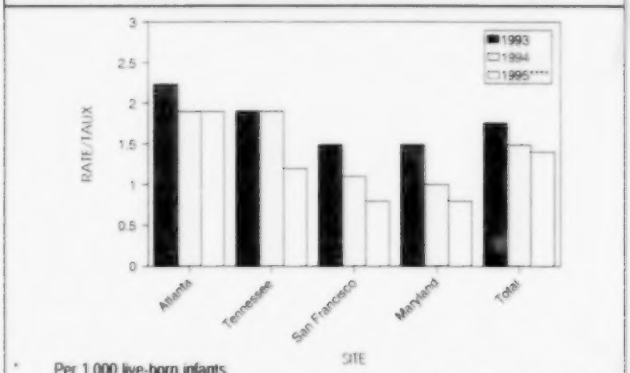
membrane rupture[4]. The overall incidence of early-onset GBS disease remained relatively constant during 1991 to 1993[5]; however, findings in this report of more recent surveillance indicate that, during 1993 to 1995, the incidence declined significantly in some areas. The recent decline may reflect the impact of adopting measures to prevent early-onset neonatal GBS infections, and improved implementation of existing clinical and laboratory policies during 1994 and early 1995.

In addition to adoption of prevention measures for early-onset GBS, other factors probably contributed to the recent decline in early-onset GBS disease. First, during 1992, the American College of Obstetricians and Gynecologists (ACOG) and the American Academy of Pediatrics (AAP) issued statements about preventing early-onset GBS disease[4]. Second, during 1994, state legislatures in California and Florida considered bills that would have mandated development and implementation of practice guidelines for preventing GBS. Although the proposed legislation was not enacted, the issues were widely publicized and resulted in state-sponsored prevention activities. Third, in 1994, CDC issued draft prevention guidelines for public comment[6] and, in 1995, provided educational materials to clinicians who had participated in previous surveys.

Because of the geographic variation in the incidence rates of GBS disease, the decline in early-onset GBS disease documented in the surveillance sites in this report may not represent national trends. However, the changes probably are not the result of surveillance artifacts because the incidence of early-onset disease declined while rates for late-onset and adult GBS disease remained stable, and because audits of microbiology laboratories have been routinely conducted in all surveillance areas. These findings are consistent with the effect of prevention efforts that can interrupt mother-to-infant transmission, rather than a change in virulence of circulating GBS strains or decreasing prevalences of GBS carriage among all age groups. Ongoing surveillance in these and additional sites is assessing whether the decline in early-onset GBS disease will continue and become more widespread.

In collaboration with ACOG, AAP, and a multidisciplinary panel of experts, CEC has developed two strategies (a screening approach and a non-screening approach) for preventing perinatal GBS disease[4,7,8]. The screening approach specifies that all pregnant women should be screened at 35 to 37 weeks' gestation for GBS carriage, and all identified carriers and women who deliver preterm before availability of a culture result should be offered intrapartum antimicrobial prophylaxis. The non-screening approach specifies that intrapartum antimicrobial agents should be offered to women with risk factors (e.g. those with elevated intrapartum temperature, membrane rupture ≥ 18 hours, or premature onset of labor or rupture of membranes at < 37 weeks). Copies of the guidelines and educational materials for prenatal patients are available from CDC's Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, Mailstop C-23, 1600 Clifton Road NE, Atlanta, GA 30333, or from the World Wide Web at http://www.cdc.gov/ncidod/diseases/bacter/strep_b.htm.

Figure 1
Incidence rate* of early-onset group B streptococcal (GBS) disease, by year and site – selected sites***, 1993-1995**



* Per 1,000 live-born infants.
** Defined as isolation of group B streptococci from a normally sterile site (e.g. blood or cerebrospinal fluid) from a resident of an area under surveillance. GBS cases were categorized as early-onset (illness onset at age < 7 days) and late-onset (illness at age 7 to 90 days).
*** The three-county San Francisco Bay area, California; four urban counties in Tennessee; the eight-county metropolitan area of Atlanta, Georgia; and the entire state of Maryland.
**** For San Francisco, Maryland, and total, $p < 0.01$, chi-square for trend.

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Source: *Morbidity and Mortality Weekly Report, Vol 46, No 21, 1997.*